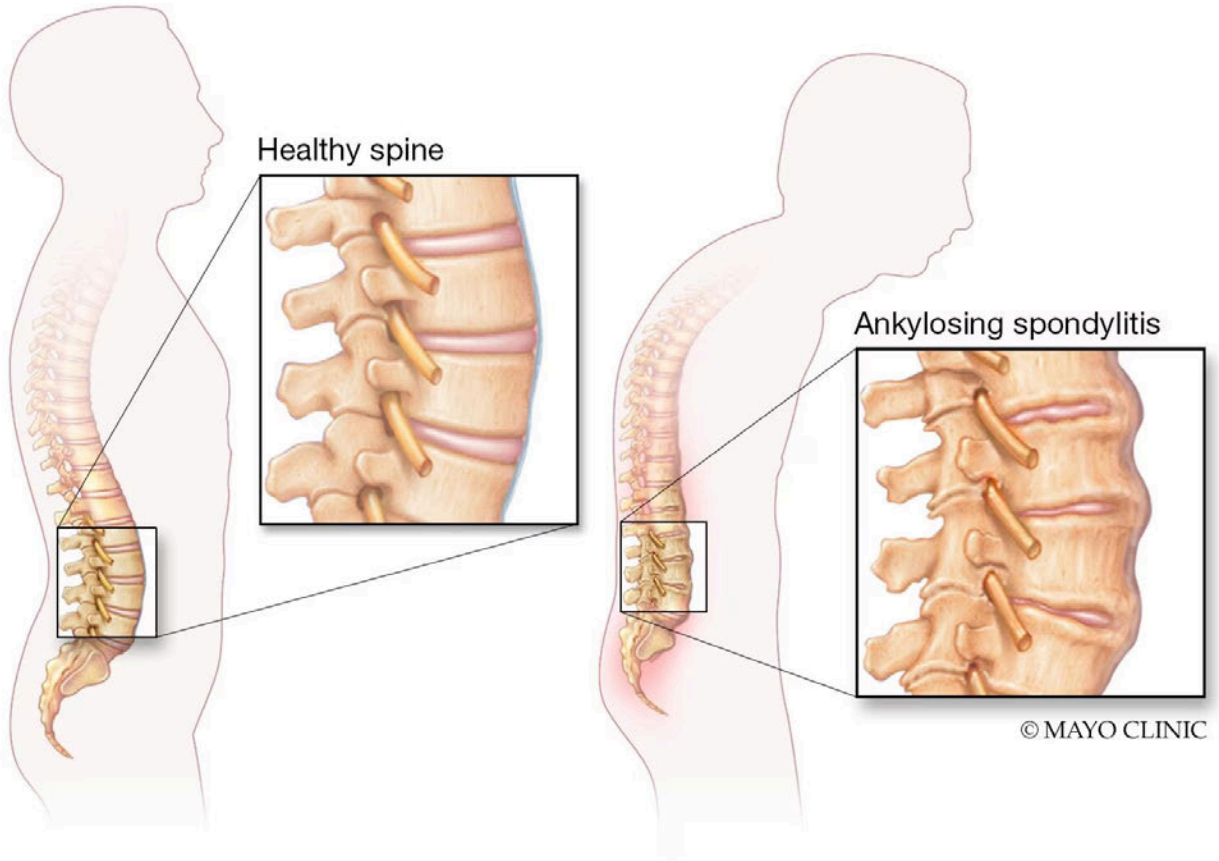
Ankylosing spondylitis

Overview

Ankylosing spondylitis, also known as axial spondyloarthritis, is an inflammatory disease that, over time, can cause some of the bones in the spine, called vertebrae, to fuse. This fusing makes the spine less flexible and can result in a hunched posture. If ribs are affected, it can be difficult to breathe deeply.



Ankylosing spondylitis

As ankylosing spondylitis worsens, new bone forms as part of the body's attempt to heal. The new bone gradually bridges the gaps between vertebrae and eventually fuses sections of vertebrae together. Fused vertebrae can flatten the natural curves of the spine, which causes an inflexible, hunched posture.

Axial spondyloarthritis has two types. When the condition is found on X-ray, it is called ankylosing spondylitis, also known as axial spondyloarthritis. When the condition can't be seen on X-ray but is found based on symptoms, blood tests and other imaging tests, it is called nonradiographic axial spondyloarthritis.

Symptoms typically begin in early adulthood. Inflammation also can occur in other parts of the body — most commonly, the eyes.

There is no cure for ankylosing spondylitis, but treatments can lessen sympto Close and possibly slow progression of the disease.

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Symptoms

Early symptoms of ankylosing spondylitis might include back pain and stiffness in the lower back and hips, especially in the morning and after periods of inactivity. Neck pain and fatigue also are common. Over time, symptoms might worsen, improve or stop at irregular intervals.

The areas most commonly affected are:

- The joint between the base of the spine and the pelvis.
- The vertebrae in the lower back.

- The places where tendons and ligaments attach to bones, mainly in the spine, but sometimes along the back of the heel.
- The cartilage between the breastbone and the ribs.
- The hip and shoulder joints.

When to see a doctor

Seek medical attention if you have low back or buttock pain that came on slowly, is worse in the morning or awakens you from your sleep in the second half of the night — particularly if this pain improves with exercise and worsens with rest. See an eye specialist immediately if you develop a painful red eye, severe light sensitivity or blurred vision.

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Causes

Ankylosing spondylitis has no known specific cause, though genetic factors seem to be involved. In particular, people who have a gene called *HLA-B27* are at a greatly increased risk of developing ankylosing spondylitis. However, only some people with the gene develop the condition.

Risk factors

Onset generally occurs in late adolescence or early adulthood. Most people who have ankylosing spondylitis have the *HLA-B27* gene. But many people who have this gene never develop ankylosing spondylitis.

Complications

In severe ankylosing spondylitis, new bone forms as part of the body's attempt to heal. This new bone gradually bridges the gap between vertebrae and eventually fuses sections of vertebrae. Those parts of the spine become stiff and inflexible. Fusion also can stiffen the rib cage, restricting lung capacity and function.

Other complications might include:

- **Eye inflammation, called uveitis.** One of the most common complications of ankylosing spondylitis, uveitis can cause rapid-onset eye pain, sensitivity to light and blurred vision. See your health care provider right away if you develop these symptoms.
- **Compression fractures.** Some people's bones weaken during the early stages of ankylosing spondylitis. Weakened vertebrae can crumple, increasing the severity of a stooped posture. Vertebral fractures can put pressure on an Close possibly injure the spinal cord and the nerves that pass through the spine.

- **Heart problems.** Ankylosing spondylitis can cause problems with the aorta, the largest artery in the body. The inflamed aorta can enlarge to the point that it distorts the shape of the aortic valve in the heart, which impairs its function. The inflammation associated with ankylosing spondylitis increases the risk of heart disease in general.

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By Mayo Clinic Staff

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The Dose–Response of Time Served in Prison on Mortality: New York State, 1989–2003

Evelyn J. Patterson, PhD

Objectives. I investigated the differential impact of the dose–response of length of stay on postprison mortality among parolees.

Methods. Using 1989–2003 New York State parole administrative data from the Bureau of Justice Statistics on state correctional facilities, I employed multinomial logistic regression analyses and formal demographic techniques that used the life table of the populations to deduce changes in life expectancy.

Results. Each additional year in prison produced a 15.6% increase in the odds of death for parolees, which translated to a 2-year decline in life expectancy for each year served in prison. The risk was highest upon release from prison and declined over time. The time to recovery, or the lowest risk level, was approximately two thirds of the time served in prison.

Conclusions. Incarceration reduces life span. Future research should investigate the pathways to this higher mortality and the possibilities of recovery. (*Am J Public Health.* 2013;103:523–528. doi:10.2105/AJPH.2012.301148)

Although several studies have documented the findings and detailed the analyses by cause of death[1,2] of postprison mortality, limited scholarship has investigated the prison environment's contribution. Several studies have shown that former prisoners are at considerable risk for drug overdose,[3–7] contributing to high mortality immediately after prison. Compared with their nonprisoner counterparts, those who have served time in prison have elevated levels of unnatural deaths. One study of persons released from prisons in Victoria, Australia, in the 1990s revealed that mortality levels owing to unnatural causes for former prisoners was twice that of prisoners and 10 times that of nonprisoners.[8] Scholarship has also found that persons who interacted with the criminal justice system were more likely to die from unnatural causes than were those who had no interaction with the criminal justice system.[9]

However, the postprison mortality literature suggests that prisoners possess characteristics coming to prison that set them apart from the rest of the population.[8–12] The selectivity of the population is an important issue but is not resolved by comparing the prison population to that of the general population or the mortality of past prisoners to people who have not been incarcerated. It is imperative to take into account that prisoners represent a small portion of those who commit delinquent acts. Prisoners are the people who were caught, indicted, and punished via incarceration. The judicial system does not capture all who commit crimes, nor are the sentencing patterns invariant across age, race, gender, and socioeconomic status.[13–18]

Some scholars have argued that the experience of incarceration can alter health trajectories because it is an axis of stratification relevant to mortality that has strong associations, as do other covariates of mortality such as race, gender, and socioeconomic status. One study showed that the experience of being incarcerated has a negative impact on life chances regardless of prior incarceration history.[19] Other work suggests a relationship between mortality and the length of time served in prison: some findings show longer stays are protective and others show the opposite.[20,21] Such studies have contributed to our understanding of the link between the criminal justice system and mortality immediately following release, focusing on mortality owing to overdose and suicide. I sought to extend this research by studying the mortality of New York State parolees over a 10-year period. By combining formal demography and survival analysis, I investigated the dose–response of time served in prison to changes in life expectancy.

METHODS

Examining the dose–response of prison requires a population that survived prison and is still under study, such as the recently paroled population. Each parolee has spent some length of time in prison and remains under the supervision of the correctional system. I used National Corrections Reporting Program administrative data, which provide comprehensive individual-level information from the state correctional facilities on persons released from parole in states that choose to submit the information. Each record includes demographic variables, variables pertaining to the offenses for which the individual was charged, variables pertaining to length of stay in prison and on parole, and type of exit from parole (e.g., revocation of parole, unconditional release, and death—the outcome of interest in this study).

I used the files pertaining to the release from parole between 1989 and 2003 for the state of New York, following the cohort of persons released from prison to parole from 1989 to 1993. This cohort of parolees was followed through 2003. Unlike the yearly data of other states for this period, New York's parole data had a low percentage of missing records and overall consistency starting in 1989.[22] New York also continued to use some type of parole, whether mandatory or discretionary, between the years 1989 and 1993. By examining the parole population, I avoided one of the main criticisms of prior studies that compared prisoners and nonprisoners: selectivity. Comparing nonprisoners to prisoners introduces issues of selectivity because persons incarcerated might be inherently different from the nonincarcerated, thereby introducing unobserved heterogeneity. However, each person who is a part of the parole population has served time in prison.

Eighty-four percent (111 509) of people released from prison in New York between 1989 and 1993 were released to parole. Table 1 displays characteristics of those who experienced an event (some type of exit from parole) during the study period. Of those experiencing an event, 2015 experienced the event of death. Although this is a retrospective study of the 1989–1993 prison release cohort that entered parole, I included 98% of the initial sample, censoring only 2%; that is, 2% of the persons in the 1989–1993 parole cohort did not experience an event by the end of 2003. The results of a sensitivity analysis investigating potential biases because of the censoring confirmed that the censoring did not influence my findings. Thus, the table of persons includes virtually the entire 1989–1993 parole cohort, and those who were censored did not affect the findings.

The sample was mostly men and mostly younger than 35.0 years. The average age at parole entrance was 30.9 years for this population. The majority of the parolees were non-Hispanic Black, followed by non-Hispanic White, and then Hispanic. These 3 groups made up 99.6% of the population experiencing an event (death, return to prison, release from parole, and all other modes). Seventy-nine percent of the sample did not complete high school. Most of the parolees were originally sentenced for drug offense, followed by violent offense, and then property offense. Almost 70.0% of the persons served 2 years or less in prison. Eight percent served 4 or more years in prison. Within 3 years of stay on parole, more than three quarters of the sample had experienced an event. I restricted my analysis to those who served 10 years or less in prison. Analyses restricting the sample to those serving 6 years or less produced similar results.

Survival Analysis

I used survival analysis to study mortality via a discrete time analysis that used maximum likelihood estimation. I broke each individual's parole history into monthly units; for each month served on parole, individuals contributed a new case. For example, an individual who served 6 months on parole contributed 6 cases. One of the strengths of hazard models is that they take into account the nonindependence of repeated observations among individuals, thereby providing unbiased estimates. Most of the variables I used—gender, race, level of education, type of crime committed, and length of stay in prison—did not vary with time and were the same for each person-month contributed to the model. Age and duration on parole, however, did vary from person-month to person-month. Most of the demographic characteristics I included controlled for base-level mortality. The type of crime committed (i.e., the most serious offense an individual was successfully charged with) operationalized risk behaviors of the individual, and the duration spent in prison accounted for the dose–response of prison.

After assembling the data into person-month units, I employed a multinomial logit model to estimate the set of simultaneous binary logits of the different modes of parole exit: death, returning to prison, release from parole, and all other modes. Thus, for each type of exit, the model produced logit coefficients that corresponded to comparing the event to the nonoccurrence of the event.

Estimating the Change in Life Expectancy

The multinomial provides the odds of the event occurring, but it is advantageous to summarize the findings in a more tangible form as well. Formal demographic methods allow estimation of the change in life expectancy because of an increase or decrease in the likelihood of death. The mean age upon entry to parole was approximately 30 years. Using the 1989–1991 US life table allows the estimation of the decrease in life expectancy at age 30 years (i.e., the number of additional or fewer years a person aged 30 years can expect to live) for each additional year lived in prison for the 1989–1993 parole cohort. A slight alteration in the Keyfitz equation[23] allows an estimation of the change in life expectancy at aged 30 years as

$$(1)\quad \Delta e(30) = -ke(30)H,$$

where H (entropy) can be calculated as

$$(2)\quad H = \frac{\int_{30}^{\infty} l(x)\ln\frac{l(x)}{l(30)}dx}{T(30)},$$

and the constant k is the change in overall mortality, which is obtained from the multinomial

TABLE 1—Composition of Persons Released From Parole: New York State, 1989–2003

Covariate	Composition (Proportion)
Gender	
Men	0.926
Women	0.074
Missing	0.001
Age, y	
≤ 24	0.258
25 29	0.275
30 34	0.218
35 44	0.193
45 64	0.054
≥ 65	0.002
Missing	0.002
Race/ethnicity	
Hispanic	0.305
Non Hispanic White	0.168
Non Hispanic Black	0.523
Other	0.004
Missing	0.007
Education	
< high school	0.790
≥ high school	0.210
Missing	0.025
Type of crime	
Violent	0.304
Property	0.224
Drug	0.393
Missing	0.000
Duration in prison, y	
< 1	0.385
1.00 1.99	0.319
2.00 2.99	0.151
3.00 3.99	0.062
≥ 4	0.083
Missing	0.000
Duration on parole, y	
< 1	0.220
1.00 1.99	0.343
2.00 2.99	0.219
3.00 3.99	0.133
≥ 4	0.085
Missing	0.000

Note. The sample size was n = 111 509.
Source. Bureau of Justice Statistics, National Corrections Reporting Program.

model. For more description of the demographic notation used in the equations, refer to a basic demographic methods text such as *Demography: Measuring and Modeling Population Processes*.[24]

RESULTS

Figure 1 shows the estimates of the hazard function of the key duration relationship, duration in prison and mortality, restricting the length of study to those who experienced an event within 10 years. The figure exhibits a relatively curvilinear relationship and increases as time served in prison increases. That is, prison had a negative dose–response on the life span.

Before estimating the multinomial logit models, I performed a Wald test to ensure the variables inserted in the models had some effect on the dependent variable. Each variable in the 3 models tested significant at the 5% level, and most tested significant at the 1% level. Table 2 displays the results of the multinomial logit for the event of death. The results for other modes of exit—returning to prison or jail, parole release, and other modes—are not shown in this table. Each model contributes to the demonstration of the relevance of time served in postprison mortality. The first model simply asserts the relationship with covariates relating to time served. The second model adds covariates to control for mechanisms known to influence mortality outcomes, and the third model considers selection issues.

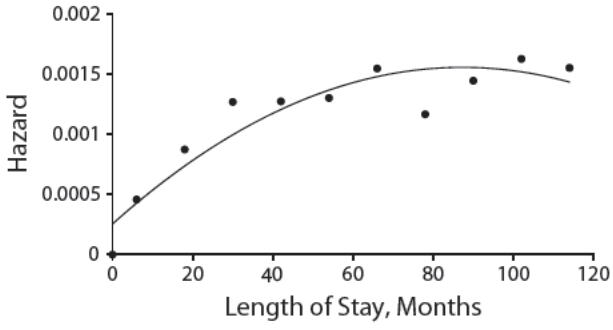
Model 1 examines the baseline model for duration in prison. The odds ratio (OR) for months in prison was 1.017. That is, for each month served in prison, the odds of dying upon release increased 1.7%, or 20.4% per year. This OR may seem small. One could argue that the odds of dying merely increased on the basis of the assumption of curvilinearity; however, when the unit of analysis was a quarter of a year or half a year, the OR increased in the expected curvilinear fashion. For example, when assuming the curvilinear increase using the OR given for 1 month, the lower bound of the confidence interval is 1.066 and the upper bound is 1.138. When the analysis is run with the unit of analysis at a quarter year and converted to half a year, the lower bound is 1.094 and the upper bound is 1.194. Finally, when the unit of analysis is half a year, the lower bound is 1.083 and the upper bound 1.167. Each of these intervals shows overlap and that the OR was not merely a function of the assumption of curvilinearity. Because of the interval and detail of observation of the data provided for the phenomena under study, I used the month as the unit of analysis.

Model 2 included several additional covariates—age, race, gender, education, and type of crime committed by the parolee. The additional covariates displayed some surprising findings, particularly with regard to ethnicity. There were stark contrasts in the level of mortality for Hispanics compared with non-Hispanic Whites. Hispanics had an odds of death that was 70% higher than that for non-Hispanic Whites, and the odds of death for non-Hispanic Blacks was 32% higher than that for non-Hispanic Whites. The gap in mortality between Hispanics and non-Hispanic Whites in the general population was not large.

After adjusting for the undercount of deaths, Smith and Bradshaw calculated the 1990 life expectancy of Hispanic women to be 79.4 compared with the 79.2 for non-Hispanic Whites, and the life expectancy of men to be 69.6 compared with non-Hispanic Whites' life expectancy of 72.8 in the general population.[25] This seems trivial compared with the 70% increased odds found in the parole population in this study. One piece of this large differential may be the housing conditions of New York's population, in which Hispanics are more likely than are Whites to rent and, when renting, more likely to live in badly maintained and overcrowded units.[26]

The findings regarding socioeconomic status went in the expected direction: those who were high school graduates had lower odds of death than did those with less than a high school education. The odds of death for women were 38% lower than were those of men. In 1990, men aged 30.00 years and living in New York could expect to live an additional 43.14 years, whereas women aged 30.00 years could expect to live an additional 49.76 years.[27] Drug offenders had lower odds of death than did those whose controlling offense was violent. This does not necessarily challenge the previous literature regarding the higher odds of death from drug overdose immediately following prison. However, it does hint at the existence of more complex interactions occurring and necessitates additional research in the area to shed light on the pathways.

In model 2, the OR for duration in prison dropped to 1.01 but remained significant. This OR means that each year in prison increased the odds of death upon exit by 12.0%. Accordingly, those spending 5 years in prison could expect a 60.0% increase in their odds of death. Part of the response to prison could be attenuated by the fact that mortality decreases as people survive parole. Perhaps as an individual survives parole, the mortality hazard function approaches the mortality levels of the general population. I tested this hypothesis in model 3 by adding covariates relating to the time spent on parole. The model shows that for each month an individual survived parole, the



Source. Calculations are from Bureau of Justice Statistics data.

FIGURE 1—Unadjusted hazard function of length of stay in prison with fitted quadratic function: New York State, 1989–2003.

TABLE 2—Multinomial Logit of Exiting Parole–Death: New York State, 1989–2003

Covariates	Model 1, OR (95% CI)	Model 2, OR (95% CI)	Model 3, OR (95% CI)
Gender			
Men (Ref)		1.000	1.000
Women		0.623** (0.503, 0.771)	0.629** (0.508, 0.778)
Race/ethnicity			
Non Hispanic White (Ref)		1.000	1.000
Hispanic		1.695** (1.458, 1.971)	1.743** (1.499, 2.027)
Non Hispanic Black		1.323** (1.144, 1.530)	1.336** (1.155, 1.544)
Other		0.298 (0.074, 1.198)	0.320 (0.080, 1.011)
Aging, mo			
Age		1.005** (1.003, 1.007)	1.006** (1.005, 1.008)
Age^2 (Ref)		1.000** (1.000, 1.000)	1.000** (1.000, 1.000)
Education			
< high school (Ref)		1.000	1.000
≥ high school		0.846** (0.754, 0.949)	0.841** (0.749, 0.944)
Type of crime			
Violent (Ref)		1.000	1.000
Property		0.988 (0.865, 1.129)	0.932 (0.816, 1.066)
Drugs		0.773** (0.691, 0.865)	0.765** (0.683, 0.856)
Other		1.057 (0.882, 1.267)	1.032 (0.861, 1.237)
Duration variables, mo			
Duration in prison	1.017** (1.001, 1.023)	1.010** (1.004, 1.016)	1.013** (1.007, 1.019)
Duration in $prison^2$	1.000** (1.000, 1.000)	1.000** (1.000, 1.000)	1.000** (1.000, 1.000)
Duration on parole			0.981** (0.974, 0.987)
Duration on $parole^2$			1.000** (1.000, 1.000)

Note. CI = confidence interval; OR = odds ratio. Other modes of exit not shown. The sample size was n = 2 774 344. 2 log likelihood = 1 011 239.
Source. Bureau of Justice Statistics, National Corrections Reporting Program.
*$P < .05$; **$P < .01$.

odds of dying decreased by approximately 2.0%. Thus, for those who survived 1 year on parole, the odds of dying decreased by 24.0%. The OR for length of stay in prison increased to 1.013 in the fully specified model. This translates to a yearly increased likelihood of death of 15.6%.

Table 3 combines the selectivity effect of surviving parole with the dose–response of prison for various lengths of stay in prison. Length of stay in this table ranged from 0 to 60 months. As the table shows, for each year served in prison, a person could expect to lose approximately 2 years of life. Thus, a person who served the mean length of stay in prison of the 1989–1993 New York parole cohort (21.7 months) could expect to live approximately 3.6 years less than could a person who did not serve time in prison. The table also lists the time to recovery or the time it takes for the person to return to the original mortality curve. The time to recovery is essentially two thirds of the time served in prison. Accordingly, those serving 6 months in prison require 4 months until their mortality curve resembles that of the general population.

DISCUSSION

The event of death, like many other health outcomes, has been shown to vary along axes of stratification such as race, ethnicity, socioeconomic status, and gender.[28–32] Much of the mortality literature consists of studies seeking to contribute to the reduction of health inequalities observed across social strata. Axes of stratification can arise because of historical or current structural inequality in the treatment of groups in addition to the behaviors and experiences of groups. A growing literature posits the experience of prison as another axis of stratification.[33–35] If incarceration is an axis of stratification, our knowledge of mortality is limited if our studies do not incorporate it when studying life outcomes. Thus, it is critical to assess the influence of incarceration on death rates, as so many other axes of stratification have proven influential in the prediction of mortality and encouraged research and actions that foster the elimination of disparities.

I examined the dose–response of prison on the mortality of parolees. Instead of focusing on differences between ex-prisoners and nonprisoners, I used the homogeneity of the parole population to ascertain information about an understudied event—the mortality of ex-prisoners as it relates to their length of stay in prison. After controlling for a variety of demographic and offense-related factors, I showed that each year in prison increased the odds of death by 15.6% in this 1989–1993 parole cohort. This translates to an increased odds of death of 78% for somebody who spent 5 years in prison and a loss of approximately 10 years in the expected life expectancy at age 30 years. The time to recovery, however, shed light on another process at work. Those who are able to survive parole without incident eventually return to the before-prison mortality curve. This finding is in line with previous research that reports an initial high risk of death at the population level that declines over time.[3,20]

Prison sentences are accompanied by a reduction in life expectancy and, thus, have a direct relationship with length of stay in prison. An increase in the odds of prison translates into real years of life lost. Although I was unable to ascertain the pathways through which this occurs, I controlled for a variety of factors that have proven important in past mortality studies. More importantly, I have made a strong case that loss of life is associated with time lived in state correctional facilities, and I have demonstrated the need for further research beyond administrative data that will permit the testing of the pathways to the higher mortality experienced and the possibility of recovery.

Limitations

My findings should be considered in the light of several limitations. First, this was an analysis of parole mortality in the state of New York.

TABLE 3—Change in Life Expectancy and Time to Recovery: New York State, 1989–2003

Length of Stay in Prison, Months	Change in Life Expectancy at Age 30 Years	Time to Recovery, Months
0	0.00	0
3	0.50	2
6	0.99	4
12	1.99	8
18	2.98	12
24	3.97	16
36	5.96	23
48	7.94	31
60	9.93	38

Source. Bureau of Justice Statistics, National Corrections Reporting Program Data.

Although New York has the largest state correctional population, other states should be studied as well as later cohorts to examine whether the health consequences of prison have changed over time. Second, the cause of death was unknown. The literature mentions the increase in particular types of mortality, and determining the differential responses of prison on natural and unnatural mortality is of interest. Third, I used the 1989–1993 parole cohort in my analyses. It is quite possible that just as mortality decreased in general, it may have also decreased for later cohorts of parolees. Fourth, I constructed event histories for persons on the basis of administrative data. Although this provides a starting place, additional variables are of interest, such as measures of social, health, and economic support of individuals on parole; better measures of risk behaviors before and after prison; and variables specific to the individual's prison experience.

This area of study demands an investigation of the variables that measure access to care. A person provided a 30-day supply of medication to manage an illness upon release from prison, for example, might not be able to connect to a new health care provider; furthermore, access to care and health benefits might prove challenging.[33,36,37]

Conclusions

Despite the study's limitations, I have indicated a new area deserving further study. Scientists have dedicated centuries of research to understanding the levels of mortality in human populations and how to lower them. I have demonstrated that one of the United States' core institutions does the opposite. This is particularly distressing considering that the United States supersedes every other nation in its propensity to incarcerate.[38]

Understanding the intended and unintended consequences of the prison experience on the lives of the incarcerated and those in their social network is a growing social issue. The lifetime likelihood of going to prison is 9.0% for men.[39] The lifetime likelihood of imprisonment in 1991 incarceration rates is 28.5% for Black men and 16.0% for Hispanic men. This translates to a prevalence rate of 12.0% for Black men and 4.9% for Hispanic men.[40] The prevalence of ever-incarcerated men grew from 3.4% in 1991 to 4.9% in 2001 (16.6% for Black men, 2.6% for White men, and 7.7% for Hispanic men).[40]

There is a growing need to understand the health consequences of incarceration because more people experience this event now than at any other moment in American history. Scholarship continues to demonstrate that punishments have a significant impact on an individual's life that continues after imprisonment.[41 45] Much work in this area of study concentrates on outcomes such as denial of citizenship rights, increased morbidity risks, and erosion of lifetime earnings and job opportunities.[44,46 50] Such collateral consequences of incarceration can be reversed. For example, advocacy groups have led many states to reinstitute the voting rights of ex-prisoners. Death, though, cannot be reversed. It is this lack of reversal that makes this area of study so consequential. ■

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Human Participation Protection

The institutional review board at Vanderbilt University approved this study.

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CRIMINOLOGY

INCARCERATION AND POPULATION HEALTH IN WEALTHY DEMOCRACIES*

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KEYWORDS: incarceration, population health, cross-national, collateral consequences

Everywhere you look, incarceration seems to be doing harm. Research has implicated incarceration not only in worse outcomes for individuals, their families, and their communities but also in growing inequality. Yet incarceration may not always harm society—even if it does harm those who experience it. To consider this possibility, I build an argument demonstrating how the macro-level consequences of incarceration may be distinctively harmful in the United States, focusing on the incarceration–health relationship as one indicator of a broader phenomenon. I then test my hypothesis by using an unbalanced panel data set including 21 developed democracies ($N = 414$) and a series of ordinary least-squares models predicting three measures of population health as a function of incarceration. Models including only a main effect of incarceration demonstrate an inverse association between changes in incarceration and changes in population health. Models including an incarceration by U.S. interaction, however, indicate that the population health consequences of changes in incarceration are far worse in the United States than elsewhere. Taken together, the results indicate that the United States is exceptional for both its rate of incarceration and its effects of incarceration, although it is unclear what drives this exceptionalism in effects.

Everywhere you look, incarceration seems to be doing harm. For the millions of Americans who spend time in prison or jail annually (Glaze and Herberman, 2013), their time behind bars on average leads to a host of negative life-course outcomes including poor labor market prospects (Pager, 2003; Western, 2002), homelessness (Geller and Curtis, 2011; Gowan, 2002), and divorce (Lopoo and Western, 2005; Massoglia, Remster,

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and King, 2011). The formerly incarcerated also suffer from a range of maladies that are partly attributable to the time that they spent behind bars. These include not just elevated mortality rates (Binswanger et al., 2007; Farrell and Marsden, 2008; Pridemore, 2014) but also both physical and mental health problems (Massoglia, 2008; Schnittker and John, 2007; Schnittker, Massoglia, and Uggen, 2012).[1]

Looking beyond the effects on inmates, the families of the incarcerated must cope with the stigma of having a family member incarcerated (Braman, 2004, Comfort, 2007, 2008) and with the often-dramatic reductions in income (Geller, Garfinkel, and Western, 2011) and increases in both economic hardship (Schwartz-Soicher, Geller, and Garinfkel, 2011) and housing instability (Geller and Franklin, 2014; Wildeman, 2014) that come along with this incarceration. Because of these effects, the women (Lee et al., 2014; Wildeman, Schnittker, and Turney, 2012) and children (Geller et al., 2012; Haskins, 2014; Roettger and Swisher, 2011; Wildeman, 2010) tied to the incarcerated tend to fare worse than expected in a whole host of important domains. Although empirical tests of the broader effects of incarceration on community life have been lacking, some research has linked living in a high-incarceration neighborhood with a host of negative outcomes, including far higher than predicted rates of both crime (Clear, 2007, 2008; Clear et al., 2003) and psychiatric morbidity (Hatzenbuehler et al., 2015).

When these effects are combined with high rates of incarceration—and racial inequality in incarceration—in the United States, the effects of incarceration may even be observable at the population level (Western and Muller, 2013; Wildeman and Muller, 2012), provided the effects of incarceration for inmates, families, and communities outweigh its crime-fighting benefits.[2] And indeed, studies testing the effects of incarceration in the United States have shown that incarceration explains much of the racial gap in AIDS (Johnson and Raphael, 2009) and some of the gaps in earnings (Western, 2006), marriage (Lopoo and Western, 2005), child well-being (Wakefield and Wildeman, 2011, 2014), and population health (Wildeman, 2012a, 2012b).

Virtually all existing research on the macro-level consequences of incarceration has to date focused solely on the United States (but see Stuckler et al., 2008),[3] however, which is problematic because there are several reasons to expect changes in the incarceration rate in the United States and in other developed democracies to yield qualitatively different results. First, and maybe most importantly, because of their low starting rates of incarceration, the crime-fighting benefits of incarceration in other developed democracies

1. For reviews of these literatures, see Massoglia and Pridemore (2015) and Wakefield and Uggen (2010).
2. Although the magnitude of the crime-fighting benefits of incarceration has long been contested (Becsi, 1999; Johnson and Raphael, 2012; Levitt, 1996; Liedka, Piehl, and Useem, 2006; Marvell and Moody, 1994; Spelman, 2008; Western, 2006), there is emerging consensus that the crime-fighting benefits of incarceration were substantial at the beginning of the prison boom but have dropped off since the early 1990s (Travis, Western, and Redburn, 2014).
3. Cross-national research has shown that including inmates in labor force calculations leads the American employment advantage relative to other developed democracies to erode (Pettit, 2012; Western, 2006; Western and Beckett, 1999). There is also some individual-level evidence, much of which uses British (Murray and Farrington, 2005, 2008) or Danish (Andersen and Wildeman, 2014; Wildeman et al., 2014) data, that parental incarceration has harmful effects on children in other developed democracies, so although there have been few macro-level studies on the effects of shifts in incarceration for macro-level outcomes, there has been a strong individual-level research base in some countries.

should still be large (Johnson and Raphael, 2012), and the negative spillover effects of increases in incarceration to communities (Clear, 2007, 2008; Clear et al., 2003) and budgets (Ellwood and Guetzkow, 2009) should still be minimal. Second, annual increases in incarceration have also been greater in the United States than they have been in other countries, and such yearly changes rather than the starting rate of incarceration may be responsible for more deleterious consequences in the United States than elsewhere. Third, conditions of confinement might be worse in the United States than in other developed democracies (Pratt, 2008), leading to differential effects. Finally, the U.S. welfare system may be less equipped to deal with a large currently and formerly incarcerated population, prompting more negative consequences. Yet despite many reasons to expect differential macro-level consequences of incarceration in the United States and other developed democracies, no research has yet tested for differential effects on any key outcomes.

In this article, I begin to fill this gap by providing the first test of the hypothesis that the population health consequences of incarceration are fundamentally larger in the United States than elsewhere. I focus on how changes in incarceration shape changes in population health by using three key dependent variables—life expectancy at birth, the infant mortality rate, and age-specific mortality rates—as indicators. These measures, and the emphasis on *population health*, are appropriate for several reasons. First, population health is generally considered to provide an excellent gauge not only of the mortality experience of a society but also of its well-being (Beckfield, 2004; Conley and Springer, 2001; Hall and Lamont, 2009). Second, the health of Americans has declined relative to the residents of comparable nations in recent decades (Berkman, 2009; Oeppen and Vaupel, 2002). Finally, because life expectancy at birth represents the cumulative mortality experience of a population, it can easily be broken into its constituent parts—age-specific mortality rates. This allows me to test for implausible effects, as well as to see whether the consequences of incarceration for mortality are concentrated in the expected age-by-sex groups. In testing for effects of incarceration on population health, this article fills an important gap in the incarceration literature. It also extends discussion of the causes of the comparatively high mortality risk of Americans relative to the residents of other wealthy democracies beyond the welfare state (Conley and Springer, 2001), income inequality (Wilkinson and Pickett, 2009), and health behaviors (Mokdad et al., 2004).

To test whether the incarceration–population-health association differs in the United States and other developed democracies, I use an unbalanced panel data set of 21 developed democracies. Although both the short-term (Wildeman, 2012a) and long-term (Johnson and Raphael, 2009) consequences of incarceration are of interest, the analyses in this article focus solely on estimating the short-term effects of incarceration. The results from these analyses demonstrate that the detrimental consequences of increases in incarceration for population health are more pronounced in the United States than in the other countries considered, although it is unclear whether these distinctive consequences are driven by the higher starting rate of incarceration in the United States, greater annual increases in incarceration, worse conditions of confinement, less developed welfare state, or some combination thereof.

INCARCERATION AND POPULATION HEALTH

Incarceration could affect population health through a whole host of channels, both direct and indirect. In this section, I highlight five channels through which incarceration

could affect population health by focusing first on three channels through which I expect to find relatively uniform effects of incarceration across developed democracies before moving on to two broader sets of mechanisms through which I expect to see differential associations across countries. Although there is a vast literature on incarceration and health (Fazel and Baillargeon, 2011; Massoglia and Pridemore, 2015), I focus on the mortality consequences of incarceration when possible, moving to broader health indicators only for the spillover effects of incarceration.

UNIVERSAL POPULATION-HEALTH CONSEQUENCES OF INCARCERATION

A tremendous amount of research has documented the first two mechanisms I consider here—that being incarcerated decreases mortality risk by dramatically cutting the risk of mortality resulting from homicide, accidents, and overdose and that being released from confinement increases mortality risk by abruptly returning further-stigmatized individuals to society with little assistance—and so I will review them briefly. Although causal evidence regarding the mortality-suppressing effects of current incarceration and the mortality-inducing effects of release is scarce (but see Pridemore, 2014), research consistently has shown that inmates die at a far lower rate than do demographically similar adults (Ginder and Noonan, 2014; Mumola, 2007; Patterson, 2010; Rosen, Wohl, and Schoenbach, 2011; Spaulding et al., 2011; for recent reviews, see Fazel and Baillargeon, 2011; Massoglia and Pridemore, 2015) and that former inmates die at a far higher rate than do demographically similar adults, especially immediately after release (Bingswanger et al., 2007; Farrell and Marsden, 2007; Rosen, Schoenbach, and Wohl, 2008; Spaulding et al., 2011; see also Loeffler, 2013; Massoglia et al., 2014; for recent reviews, see Fazel and Baillargeon, 2011; Massoglia and Pridemore, 2015). Because of how strong and universal these relationships seem to be, it would be surprising if all developed democracies did not experience increases in incarceration tied with lower mortality rates among the men in the ages where the largest portion of their mortality was a result of preventable causes of death and with higher mortality rates among the men in the ages where permanent prison and jail release is most common.

The incarceration of young men also could reduce the mortality risk of demographically similar individuals with whom they might have been in violent encounters had they not been incarcerated,[4] which represents a third possible universal linkage between incarceration and population mortality. Such indirect effects of incarceration on mortality are difficult to isolate, but some research based on the United States has presented supportive evidence (Wildeman, 2012a: 85). The magnitude of these indirect mortality-reducing benefits of incarceration could vary across countries, but it seems reasonable to expect the noted protective effects in most settings.

4. For a discussion of the overlap between homicide victims and perpetrators, see Papachristos (2009).

CONTEXTUAL CONSEQUENCES OF INCARCERATION FOR POPULATION HEALTH

Although there are several ways in which the incarceration–population-health association should be universal—or at least close to universal—there are also several ways in which incarceration could have differential effects on population health in the United States and in other countries. In this regard, one especially important possibility is the health of infant children. In the United States, research has shown that having a parent incarcerated is associated with an increased risk of infant mortality and that increases in imprisonment rates also lead to marked increases in the infant mortality rate (Wildeman, 2012b: 231–3). The limited evidence in the United States thus suggests that incarceration compromises the health and well-being of infants.

Yet there are reasons to expect that increases in incarceration will not promote infant mortality in other developed democracies. Although not a goal of incarceration, incarceration likely limits the short-term fertility of individuals. If those in the population who experience incarceration are most likely to have high-risk births, which seems likely given the overlapping risk factors for these events (Western, 2006; Wise, 2003), then increases in incarceration could decrease the infant mortality rate by limiting the number of high-risk births. Within the United States, where increases in incarceration are positively associated with changes in the infant mortality rate (Wildeman, 2012b), it would be reasonable to conclude that this decline in high-risk births is insufficient to offset the negative effects of having a parent incarcerated for infant's survival. Yet in countries with a more generous welfare state, it is not unreasonable to expect that incarceration may have a qualitatively different effect. Indeed, in countries with generous welfare states, household economic resources and access to medical care, two of the mechanisms proposed to link incarceration and infant mortality (Wildeman, 2012b: 231–2), may be less affected by incarceration than in the United States, buffering the individual-level consequences of incarceration for infant mortality. Under these conditions—of aggregate-level declines in high-risk births and buffered consequences of family member incarceration for individual infant's survival probability—it might even be reasonable to expect increases in the incarceration rate to lead to decreases in the infant mortality rate in other developed democracies.

The broader spillover effects of increases in incarceration on adult family members and communities that have recently received so much attention in the mostly U.S.-based literature (Hatzenbuehler et al., 2015; Johnson and Raphael, 2012; Lee et al., 2014, 2015; Wildeman, Schnittker, and Turney, 2012) may also be contextually dependent. Although much of this research has suggested that the effects of increases in incarceration are shared equally by males and females (Hatzenbuehler et al., 2015; Johnson and Raphael, 2012), research has also suggested that having a family member incarcerated puts women, but not men, at elevated risk for a host of risk factors for cardiovascular disease (Lee et al., 2014), implying that the broader spillover effects of high rates of incarceration for the health of women may be greater than the effects for men.

Virtually all research on the community-level consequences of incarceration either has used highly disadvantaged samples in which the average starting rate of incarceration was high (Hatzenbuehler et al., 2015) or has assumed that community-level incarceration rates only have significant harmful effects when incarceration is highly prevalent (Clear, 2008), and as such, it is unclear how these results generalize to other contexts with

lower incarceration rates. Because no other countries are likely to have community-level incarceration rates anywhere near the tipping point hypothesized within the United States, it would be reasonable to assume small, even negligible, spillover effects in this regard outside of the United States. This is especially the case given the consequences of exposure to crime for individuals (Sharkey et al., 2012). The same is likely the case for spillover effects on family members as countries with a more highly developed welfare state, a lower starting rate of incarceration, or a more modest annual increase in incarceration may be better able to minimize the family consequences of incarceration.

Thus, the little existing research on incarceration and population health has implied the possibility of some contextually dependent consequences of incarceration for population health, with the limited evidence suggesting that the broader spillover effects of incarceration should be most negative in the United States and should be primarily null in other developed democracies.

PREDICTIONS

Based on existing research, I have two core predictions. First, I expect changes in incarceration to be inversely associated with changes in population health. Second, I expect increases in incarceration to have worse consequences for population health in the United States than in other developed democracies. Because I will test both of these core predictions only on the broadest measures of population health (life expectancy and infant mortality), I also have three predictions involving the relationship between changes in incarceration and changes in age- and sex-specific mortality rates. First, increases in incarceration will be associated with decreases in mortality among young men and with increases in mortality among men closer to the average age of prison release in both the United States and the other 20 developed democracies considered here. Second, increases in incarceration will only be associated with increases in female mortality rates in the United States as it is only in the United States that I expect to see significant spillover effects of incarceration on mortality (and female incarceration rates are likely too low to have an effect on aggregate mortality rates). Third, increases in incarceration will only be associated with significant increases in mortality for infants, children, and older persons in the United States. These three predictions revolving around age- and sex-specific effects of incarceration on mortality provide a vital plausibility check for the stage of the analysis focusing on broader measures.

DATA AND ANALYTIC STRATEGY

DATA

I use an unbalanced panel data set covering the years 1981–2007 ($N = 414$) and including 21 countries that were either founding members of the Organisation for Economic Co-operation and Development (OECD) or wealthy democracies at the beginning of the period. The data were drawn from various sources (table A1 in appendix A in the online supporting information[5]).

5. Additional supporting information can be found in the listing for this article in the Wiley Online Library at http://onlinelibrary.wiley.com/doi/10.1111/crim.2016.54.issue-2/issuetoc.

Table 1. Descriptive Statistics ($N = 414$)

Variable	Mean	(SD)	Min	Max
Dependent Variables				
Total life expectancy at birth (in years)	77.7	(1.8)	73.6	82.4
Male life expectancy at birth (in years)	74.7	(2.0)	70.2	79.2
Female life expectancy at birth (in years)	80.7	(1.7)	76.4	85.8
Infant mortality rate (per 1,000)	5.8	(1.8)	2.4	11.4
Explanatory Variable				
Incarceration rate in previous year (per 1,000)	1.1	(1.1)	.3	7.4
Control Variables				
Total fertility rate	1.7	(.2)	1.2	2.4
Percent of the population 65+	14.4	(2.2)	9.6	20.8
Per capita GDP in 2000 dollars (logged)	10.1	(.2)	9.5	10.8
Unemployment rate	7.3	(3.9)	.5	23.9
Per capita public health care expenditures in 2000 dollars (logged)	7.4	(.3)	6.5	8.0
Per capita social expenditures in 2000 dollars (logged)	8.6	(.3)	7.5	9.5
Income inequality (Gini)	28.9	(4.2)	20.9	37.9
Homicide rate (per 100,000)	1.9	(1.7)	.2	9.8

ABBREVIATIONS: GDP = gross domestic product; SD = standard deviation.
Source: Table A1 in online supporting information.

Dependent Variables

I use three sets of dependent variables: measures of life expectancy at birth, infant mortality rates, and age-specific mortality rates. Life expectancy at birth provides an overview of the health of a population (see especially Hall and Lamont, 2009; see also Beckfield, 2004). As the associations between incarceration and health may vary by sex, I consider not only total life expectancy but also male and female life expectancies. The infant mortality rate is an indicator of the health of women of childbearing age and their infants (Conley and Springer, 2001). The final dependent variables are age-specific mortality rates for males and females. I break age-specific mortality rates into 5-year age groups (to 65–69), with the exception of considering 0–1 and 1–4 as separate categories. I consider age-specific effects of incarceration on mortality because it allows me to 1) detect implausible effects of incarceration on mortality rates and 2) isolate age-specific variations in the mortality effects of incarceration.

Before moving on, it is worth noting that the measures of life expectancy for some countries are based on a 3-year moving average (Australia, Canada, and New Zealand; OECD, 2015). Because these measures were not based on a single year, I also ran analyses using a 2-year lagged incarceration rate and a 3-year lagged incarceration rate. These analyses, which are available upon request, yielded substantively identical conclusions, implying the differences across countries in the measurement of life expectancy is not influencing my results.

Table 1 presents descriptive statistics on life expectancy, infant mortality, and the controls. Average life expectancy is 77.7 years, with females (at 80.7 years) expected to live 6 years longer than males (at 74.7 years). The average infant mortality rate was 5.8 per 1,000. Table S1 in the online supporting information presents descriptive statistics on age-specific mortality rates.

Figure A1 in appendix A in the online supporting information shows how life expectancy at birth increased across these developed democracies. All countries included in the analysis experienced an increase in life expectancy at birth over this period,

although there were pronounced variations across countries in how much their life expectancy increased. The average increase was .22 years of life expectancy annually, with the largest increase taking place in New Zealand (.29 years annually) and the smallest ones taking place in the Netherlands (.15 years annually) and the United States (.16 years annually).

Figure A2 in appendix A in the online supporting information shows a similar pattern toward increasing population health over this period by showing the reductions in the infant mortality rate. The average annual decline in the infant mortality rates in these countries was .22 per 1,000, with Japan experiencing the smallest decrease annually at .13 per 1,000 (although from a low starting infant mortality rate of approximately 5.20 per 1,000) and Portugal experiencing the largest decrease annually at .47 per 1,000.

Explanatory Variable

The explanatory variable is the incarceration rate in the previous year and is measured per 1,000 individuals in the population. I use the incarceration rate, which includes prison and jail confinement, rather than the imprisonment rate, which includes only prison confinement, because it is easier to compare across nations than the imprisonment rate is. I focus on the total rate because sex-specific estimates of the incarceration rate are available for too few country-years to provide a strong test of sex-specific effects (Walmsley, 2012).

By using the incarceration rate in the prior year to predict population health in the current year, I provide insight only into the *immediate* consequences of incarceration for population-level mortality, and as such, the results presented in this article do not capture the longer term mortality consequences of incarceration that work indirectly through channels ranging from elevated AIDS infection rates (Johnson and Raphael, 2009) to higher rates of mental and physical health problems as a result of incarceration (Hatzenbuehler et al., 2015; Lee et al., 2014). I focus on these short-run effects because a complete analysis of the long-run consequences of incarceration for mortality, which could include a long series of lags for some causes of death (Johnson and Raphael, 2009), would not produce stable estimates given the small number of observations I have for each country. Nonetheless, a more complete analysis of this relationship, including a long series of lags, is an especially pressing task for future research.

As table 1 indicates, the average incarceration rate per 1,000 was 1.1 in the analytic sample. In figure A3 in appendix A in the online supporting information, which shows trends in the incarceration rate across countries over these years (in the traditional metric of per 100,000), the United States stands out as an extreme outlier. The average annual increase in the incarceration rate over this period was 1.6 inmates. The average annual increase for the United States, however, was 23.0 inmates. The country with the next largest average increase (New Zealand) added approximately 3.8 inmates per 100,000 annually.

Control Variables

This analysis also includes a host of control variables. These include the total fertility rate, the percentage of the population 65 years of age or older, per capita gross domestic product (GDP), the unemployment rate, public expenditures on health, total social expenditures, income inequality, and the homicide rate. Until recently, data on income

inequality were limited, making it difficult to control for income inequality in models using repeated observations on countries without losing many observations. However, a new data set called "Standardizing the World Income Inequality Database" (SWIID) provides information on income inequality for all country-years included in this analysis (Solt, 2009). Changes in incarceration do not correspond closely with changes in crime in the United States in this period (Western, 2006), but I still control for the homicide rate. I do so for three reasons. First, homicide is also a cause of death. Second, research has suggested a substantial association between incarceration and homicide rates in other countries (Nadanovsky and Cunha-Cruz, 2009). Finally, homicide is the crime most unlikely to be defined dramatically differently across countries.[6]

Although the tables presented in the body of this article suppress the coefficients for the controls, in the interest of parsimony, table A2 in appendix A in the online supporting information shows the full results for the life expectancy and infant mortality models, including all coefficients for the control variables.

ANALYTIC STRATEGY

For all analyses, I rely on an ordinary least-squares (OLS) regression model with country and year fixed effects and standard errors based on the Huber–White sandwich estimator. This model is appropriate because it controls for stable (yet unobserved) traits of countries possibly associated with both incarceration rates and population health, as well as dealing with the possibility that yearly variations in incarceration and population health may be better modeled with a series of dummy variables than with a smaller number of terms capturing this variation.[7] In cross-national analyses, unobserved heterogeneity is among the most serious threats to causal inference, so including country fixed effects improves the reliability of results, although it is worth pointing out that a model estimating the incarceration–population-health association on the basis of exogenous variation in incarceration would be preferable. Unfortunately, isolating exogenous variation in incarceration, whether within the U.S. context (Levitt, 1996) or the international one (Drago, Galbiati, and Vertova, 2009) is difficult, and hence, this analysis is not able to do so. This omission is problematic because it leaves open for debate whether any association between incarceration and population health is causal or is instead driven by other channels. As such, future research might consider testing whether the method used by Johnson and Raphael (2012) to isolate exogenous variation in incarceration could be extended internationally.

6. Given research showing that political constellations influence imprisonment rates, it would have been reasonable to control for such measures. Following Sutton (2000: 380), some analyses initially included a control for right-party dominance. These analyses produced almost identical results for all analyses included in this article but resulted in the loss of 157 country-year observations, representing 38 percent of the initial observations, so I excluded this control.
7. Although a feasible generalized least-squares (FGLS) model with random and fixed effects would have improved efficiency, I did not use that model because Hausman tests revealed significant differences between the FGLS and OLS estimates. The efficiency gained from using random effects is preferable to fixed effects only when it does not significantly alter the coefficients (Halaby, 2004; see also Beckfield, 2006), so I use the OLS regression model with fixed effects. Nonetheless, estimated incarceration–population-health associations were similar in FGLS models with fixed and random effects.

At least two other related difficulties stem from the use of a panel data, however, both of which could lead to biased standard errors, leading me to over- or underreject the null hypothesis: 1) the clustering of observations on countries and 2) serial correlation. To deal with these two issues, I include country and year fixed effects and use standard errors based on the Huber–White sandwich estimator. I opt for these standard errors for two reasons. First, because most models that deal with serial correlation assume an autoregression [AR(1)] process for the serial correlation, they misestimate standard errors when the process is not AR(1), which is often the case (Bertrand, Duflo, and Mullainathan, 2004: 272). Second, although research often has suggested using cluster-robust standard errors for balanced panels with a large number of clusters (Cameron and Miller, 2015), when the number of clusters is small[8] in an unbalanced panel, cluster-robust standard errors do not perform as well as they do with a larger number of clusters or in a balanced panel (Cameron, Gelbasch, and Miller, 2008: 421–2) and hence are likely inappropriate. For the main results presented in this article, I therefore use standard errors based on the Huber–White sandwich estimator. But I also show in table S2 in the online supporting information how the *p* values change across the life expectancy and infant mortality models when an OLS model with 1) default standard errors or 2) with cluster-robust standard errors or 3) a feasible generalized least-squares (FGLS) model with default standard errors assuming an AR(1) process is used instead. Although the cluster-robust standard errors always lead to larger *p* values, the results are nonetheless highly consistent regardless of which combination of model type and standard error type I is selected.

Stationarity is also a concern as failing to address this issue can lead to biased estimates (Spelman, 2008). Because results from cross-sectionally augmented Dickey–Fuller tests (on the full panel and the strongly balanced panel) and the Hadri LaGrange Multiplier test (on the strongly balanced panel) did not provide definitive evidence regarding the stationarity of the 49 dependent variables (total life expectancy, female life expectancy, male life expectancy, infant mortality, 15 total age-specific mortality rates, 15 female age-specific mortality rates, and 15 male age-specific mortality rates), it was unclear whether the modeling strategy applied in this article or a first difference strategy would be more appropriate. After strongly balancing the panel to include only countries with complete data from 1991 to 2005, which involved dropping 144 observations (representing 35 percent of the country-years included in the complete analysis), results were similar using a first difference model.[9]

The analysis proceeds in four stages. In the first stage (table 2, models 1–4), I consider the association between incarceration and population. These models specify this relationship by using only a main effect of incarceration that applies to all 21 countries considered in this article.

8. Although there is no hard-and-fast rule for what constitutes a relatively small number of clusters, most researchers in this area would suggest that less than 50 clusters is relatively small, and nearly all would suggest that less than 20 clusters is relatively small (Cameron, Gelbasch, and Miller, 2008; Cameron and Miller, 2015). I have 21 clusters.
9. The only exception is that the relationship between incarceration and the infant mortality rate in the full sample (reported in table 2) is not statistically significant in the first difference model. It is unclear whether this difference is attributable to the change in the analytic sample or to the different modeling strategy.

Table 2. Results from OLS Regression Models Predicting Population Health Measures as a Function of Incarceration, 1981–2007

	Main Effects Only								Including an Incarceration by United States Interaction							
Variable	**M1 (Total LEB)**		**M2 (Male LEB)**		**M3 (Female LEB)**		**M4 (IMR)**		**M5 (Total LEB)**		**M6 (Male LEB)**		**M7 (Female LEB)**		**M8 (IMR)**	
Incarceration	−.33***	(.04)	−.29***	(.05)	−.37***	(.04)	−.25*	(.10)	.12	(.17)	.23	(.17)	−.01	(.20)	−1.19***	(.28)
Incarceration × U.S.	–	–	–	–	–	–	–	–	−.49**	(.18)	−.57**	(.18)	−.39†	(.21)	1.56***	(.26)
Country FE	YES		YES		YES		YES		YES		YES		YES		YES	
Year FE	YES		YES		YES		YES		YES		YES		YES		YES	
Controls	YES		YES		YES		YES		YES		YES		YES		YES	
R^2	.98		.98		.98		.94		.98		.98		.98		.94	
N	414		414		414		414		414		414		414		414	

NOTES: Standard errors are in parentheses. All t tests are two-sided. All analyses use standard errors based on the Huber–White sandwich estimator. For the full set of results, including coefficients for all controls and the intercept, see table A2 in online supporting information.
ABBREVIATIONS: FE = fixed effects; IMR = infant mortality rate; LEB = life expectancy at birth.
†$p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed).

Figure 1. Estimating the Magnitude of the Effect of Changes in Incarceration on Life Expectancy at Birth

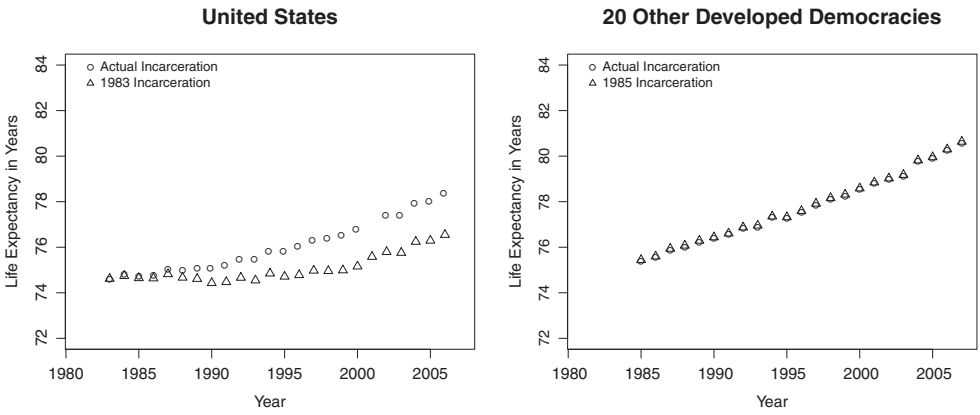
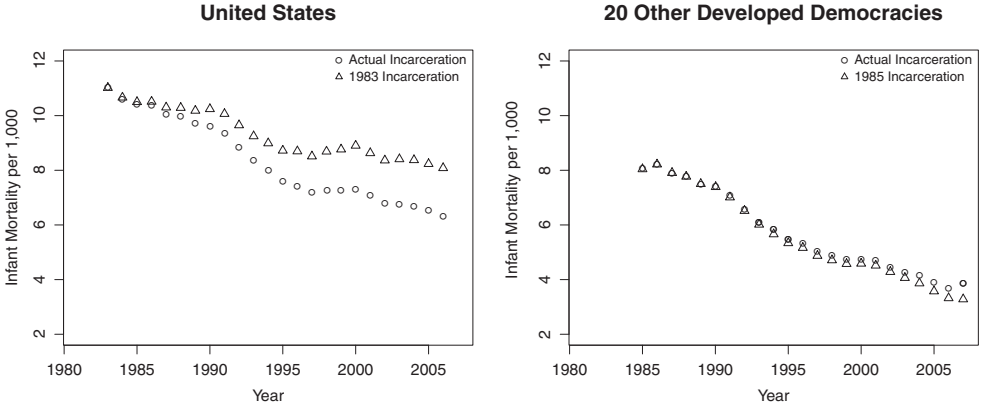


Figure 2. Estimating the Magnitude of the Effect of Changes in Incarceration on Infant Mortality



In the second stage (table 2, models 5–8), I model an interaction between the United States and the incarceration rate in my models predicting life expectancy at birth and the infant mortality rate.

In the third stage (figures 1 and 2), I show how large these effects are at the macro level by predicting life expectancy and the infant mortality rate for countries based on 1) the model including only a main effect and 2) the model including the interaction effect under the counterfactual scenario where the country's incarceration rate had remained at its starting level.

In the final stage (table 3), I consider the association between incarceration and age-specific mortality rates. Unlike the other stages of the analysis, which rely on an interaction term to model differential effects, this stage relies on models including the United States and models excluding the United States to do so. The models excluding the United States are an especially important check of the results from stage two as they allow me

Table 3. Age-Specific Mortality Effects of Incarceration, 1981–2007

Age	Total	Male	Female
Including the U.S. ($N = 414$)			
<1	.000249*	.000308**	.000200*
	(.000097)	(.000117)	(.000093)
1–4	.000007	−.000009	.000020*
	(.000008)	(.000011)	(.000008)
5–9	.000012**	.000011*	.000011*
	(.000004)	(.000005)	(.000004)
10–14	.000003	.000008	−.000005
	(.000005)	(.000007)	(.000005)
15–19	.000027*	.000034	.000000
	(.000012)	(.000018)	(.000006)
20–24	.000056*	.000062*	.000017*
	(.000017)	(.000024)	(.000009)
25–29	.000047*	.000031	.000032**
	(.000020)	(.000031)	(.000010)
30–34	.000046*	.000023	.000034***
	(.000019)	(.000030)	(.000008)
35–39	.000108***	.000076**	.000096***
	(.000017)	(.000024)	(.000012)
40–44	.000171***	.000171***	.000110***
	(.000021)	(.000029)	(.000018)
45–49	.000169***	.000139***	.000100***
	(.000028)	(.000029)	(.000028)
50–54	.000189***	.000168***	.000050
	(.000045)	(.000046)	(.000042)
55–59	.000366***	.000363***	.000109*
	(.000064)	(.000065)	(.000045)
60–64	.000659***	.000647***	.000278***
	(.000099)	(.000106)	(.000065)
65–69	.001054***	.000873***	.000671***
	(.000159)	(.000179)	(.000098)
Excluding the U.S. ($N = 390$)			
<1	−.001228***	−.001464***	−.001029***
	(.000287)	(.000361)	(.000287)
1–4	−.000061**	−.000099**	−.000016
	(.000023)	(.000038)	(.000029)
5–9	−.000011	−.000014	−.000004
	(.000012)	(.000018)	(.000016)
10–14	−.000042**	−.000024	−.000059*
	(.000016)	(.000027)	(.000021)
15–19	−.000141***	−.000229***	−.000031
	(.000031)	(.000049)	(.000027)
20–24	−.000160***	−.000266***	−.000028
	(.00004)	(.000062)	(.000009)
25–29	−.000011	−.000035	.000014
	(.000044)	(.000081)	(.000039)
30–34	.000172**	.000314***	.000015
	(.000055)	(.000087)	(.000032)
35–39	.000320***	.000477***	.000145***
	(.000046)	(.000067)	(.000045)
40–44	.000214***	.000398***	.000036
	(.000056)	(.000084)	(.000052)
45–49	−.000066	−.000003	−.000091
	(.000069)	(.000088)	(.000089)
50–54	−.000260†	−.000216	−.000200
	(.000148)	(.000172)	(.000168)

(*Continued*)

Table 3. Continued

Age	Total	Male	Female
55–59	−.000235	−.000018	−.000260
	(.000195)	(.000267)	(.000172)
60–64	−.000134	−.000116	−.000058
	(.000314)	(.000437)	(.000288)
65–69	−.000501	−.000969	−.000181
	(.000490)	(.000669)	(.000419)

NOTES: All models include country FE, year FE, and all controls listed in table 1. Standard errors are in parentheses. All *t* tests are two-sided.
ABBREVIATION: FE = fixed effects.
$^{\dagger}p < .10$; $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$ (two-tailed).

to check for implausible effects, which would suggest a spurious relationship not noticed earlier, as well as to see whether the consequences of incarceration for mortality are concentrated in the ages that might be expected based on existing research.

RESULTS

RESULTS FROM MODELS ESTIMATING ONLY THE MAIN EFFECT OF INCARCERATION

In models 1–4 of table 2, I present estimates of the association between the incarceration rate and two measures of population health, life expectancy at birth and the infant mortality rate, in 21 wealthy democracies over the 1981–2007 period. I also present sex-specific results for life expectancy at birth. All models include country fixed effects, year fixed effects, and all controls.

These models paint a consistent portrait of increases in incarceration being inversely associated with improvements in population health, which is in line with previous research on the population health implications of incarceration in the United States (Wildeman, 2012a, 2012b). For life expectancy, the estimated effect suggests that each 1 per 1,000 increase in incarceration is associated with a .29 (for males) to .37 (for females) year decline[10] in life expectancy at birth, with both associations being statistically significant at the .001 level. (The association with total life expectancy is similar.) The association between incarceration and the infant mortality rate is only significant at the .05 level, but this association nonetheless indicates a substantial effect, with each 1 per 1,000 increase in incarceration tied to a .25 per 1,000 increase in infant mortality. Thus, the results from this stage of the analysis show a strong, consistent main effect.

RESULTS FROM MODELS THAT ALSO INCLUDE AN INTERACTION EFFECT

Models 1–4 in table 2 suggest an inverse association between increases in incarceration and changes in population health in developed democracies, but since the United

10. It is important to keep in mind, as figures A1 and A2 in appendix A in online supporting information show, that all nations experienced increases in both life expectancy and infant mortality over this time. As such, when I discuss incarceration leading to declines in population health, I only mean to indicate declines in the population health gains experienced over this period.

States is such an outlier in terms of incarceration and there are reasons to expect the incarceration–population-health association to differ in the United States and in other developed democracies, models that do not consider this possibility may not accurately reflect the relationship between incarceration and population health for other nations. Models 5–8 test whether the incarceration–population-health association is distinctively adverse in the United States by including an interaction term. In three of the four cases (for total life expectancy, male life expectancy, and infant mortality), there is evidence at the .01 level or better that incarceration has a significantly more adverse effect on population health in the United States. In the other cases (for female life expectancy), the evidence for such an interaction effect is weaker, although still marginally significant.

Although the standard errors for the main effects of incarceration in models 5–8 suggest notable heterogeneity in its impact across non-U.S. nations, the results indicate that the adverse effect of incarceration on mortality is much smaller for these nations as a whole than it is in the United States. The estimated effects, moreover, are substantial. For total life expectancy, for instance, each 1 per 1,000 increase in the incarceration rate is associated with a statistically insignificant increase of .12 years in all developed democracies except the United States and a .37 year decline in life expectancy in the United States. For infant mortality, each 1 per 1,000 increase in the incarceration rate is associated with a statistically significant decrease of 1.19 per 1,000 in the infant mortality rate in all developed democracies except the United States and an increase of .37 per 1,000 in the United States. Thus, these results provide strong support for a distinctive incarceration–population-health association in the United States.

HOW MUCH DOES INCARCERATION AFFECT POPULATION HEALTH IN WEALTHY DEMOCRACIES?

To gauge just how large the consequences of changes in incarceration could be for population health both in the United States and in other developed democracies, figure 1 plots total life expectancy at birth including the main effect from model 5 and the interaction effect for model 5 (which only affects the United States) under two scenarios: 1) holding the incarceration rate at its starting level and 2) allowing the incarceration rate to change as it did over the period. For both of these scenarios, I allow all other values to change as they did and generate predicted values for life expectancy to change accordingly. Thus, the "Actual Incarceration" points represent the predicted life expectancy in these 21 countries as a result of how all independent variables changed; the other predicted values represent the predicted life expectancy in these countries, assuming all other independent variables changed as they did but incarceration remained at its starting level.

Because of the modest growth in incarceration in all countries included in figure 1 except the United States and the small point estimates from model 5 in table 2 (indicating that each 1 per 1,000 increase in incarceration is associated with a statistically insignificant increase in total life expectancy at birth of .12 years), the macro-level consequences of incarceration for total life expectancy at birth were muted for the pooled sample. As a result, the two lines for the 20 developed democracies other than the United States in figure 1 are indistinguishable. This could not be further from the truth for the United States. For total life expectancy at birth, the results from figure 1 indicate that the United States missed out on 1.79 years of total life expectancy gains as a result of increases in incarceration over this period. As the observed increase in life expectancy in the United

States over this period was 3.50 years, this means that absent increases in incarceration, my model predicts that life expectancy at birth in the United States would have increased 51.10 percent more than it did. Because macro-level effects outside of the United States are minute, mass incarceration could have substantially increased inequality in life expectancy at birth between the United States and other developed democracies.

The results from figure 2, which considers infant mortality, are similar. According to these predictions, the U.S. infant mortality rate would have declined 1.78 per 1,000 more absent increases in incarceration, meaning that it would have decreased 39.60 percent more absent increases in incarceration (4.50 per 1,000 observed). For the pooled sample, macro-level effects of changes in incarceration are again small. Thus, the results from figure 2 suggest that mass incarceration has greatly increased disparities in infant mortality between the United States and other developed nations.

AGE-SPECIFIC EFFECTS OF INCARCERATION ON MORTALITY

The results presented to this point are provocative. Yet absent age- and sex-specific analyses of the mortality effects of incarceration, it remains unclear whether these results are driven through plausible channels, suggesting a real effect, or implausible ones, suggesting a spurious relationship. In table 3, I estimate age-specific associations between incarceration and mortality for the total, male, and female population to provide insight into how the relationship between incarceration and mortality is concentrated in different age groups. Before moving on from this analysis, however, it is worth reviewing what age-specific mortality effects seem plausible. First, increases in the incarceration rate will be associated with decreases in mortality among men in their late teens and early 20s and with increases in mortality among men in their 30s in all countries. Second, increases in the incarceration rate will only be associated with elevated rates of mortality for infants, children, adult women, and older persons in the United States.

For the full sample, the age-specific associations are largely consistent with what would be expected based on U.S.-specific research on mortality—and health effects more broadly. Incarceration growth does not reduce mortality among those in their late teens and early 20s for the full sample, but the other age patterns fit the expected pattern. It is unclear why the protective effects of incarceration are not found in the full sample but are in the sample excluding the United States, and this odd finding deserves greater attention in future research. Aside from this one odd finding, there is the usual increase in infant and child mortality associated with increases in the incarceration rate (Wildeman, 2012b). And there is the usual uptick in male mortality around the age at which inmate release is most concentrated (Binswanger et al., 2007). And there is also evidence of population-level mortality consequences of increases in incarceration not only for adult women, especially in their 40s, but also the entire population in the later ages, which again is highly consistent with research on the spillover effects of incarceration (Clear, 2007). Thus, the results for the full sample are broadly consistent with what would be expected on the basis of U.S.-based research, which makes sense because, as we have seen before (models 1–4 in table 2), the results for the full sample tend to be driven by the massive changes in the U.S. incarceration rate over this period.

In the second set of panels, I shift to estimating the age-specific effects of increases in the incarceration rate outside of the United States. I use this restricted sample instead of an interaction effect because focusing exclusively on these age-specific associations

in the 20 countries in which incarceration does not seem to have discernible negative consequences for population health makes it easier to detect implausible associations than it would be by using a set of main effects and interaction effects across such a large model space (results from 45 models are presented).

The results for males provide support for three key findings. First, increases in incarceration are associated with substantial and statistically significant declines in mortality for boys younger than 5 years of age, with effects concentrated among infants. Second, increases in incarceration are associated with substantial and statistically significant declines in the mortality rate of males between 15 and 24 years of age—precisely the age when preventable forms of mortality such as homicide, overdose, and accidents could be diminished through incapacitation. Finally, increases in incarceration are associated with significant increases in the mortality rates of males between 30 and 44 years of age—the age range in which individuals who have been imprisoned would be likely to be released and to be at the elevated risk of mortality that comes along with that event. Thus, age-specific incarceration–mortality associations for males are highly consistent with what the existing literature would suggest in terms of the pros and cons of incarceration for mortality.

The results from analyses considering age-specific associations between incarceration and population health for females in countries with low rates of incarceration also support the idea that the relationship between incarceration and population health in these countries is null. The key difference between these results and the results for males, however, is that these results provide support for my hypotheses by showing the null effects of changes in the incarceration rate across the board for adult females, with the sole exception being females 35–39 years of age, which is consistent with the idea that because few women experience incarceration in these countries (Walmsley, 2012) and the spillover effects of incarceration should be minimal outside of the United States, it would be surprising to observe any effects of changes in the incarceration rate on female mortality.

DISCUSSION, IMPLICATIONS, AND LIMITATIONS

The results from this analysis, which considered the relationship between incarceration and population health in 21 developed democracies, support three conclusions. First, in models including only a main effect, increases in incarceration are inversely associated with gains in population health. Second, when a United States by incarceration interaction effect is included in the models, the association between incarceration and population health changes dramatically. Incarceration is no longer linked with a significant decline in life expectancy for the rest of the countries, and increases in incarceration are linked with significant and substantial declines in infant mortality, suggesting a protective effect of incarceration on population health. These differential effects, moreover, are significant at the conventional .05 level for three of the four outcomes considered here. And they are substantial—especially for the infant mortality rate—indicating that increases in the incarceration rate in the United States over the last 25 years may have done even more to push the United States to the back of the pack in terms of population health than the models including only a main effect of incarceration imply. Finally, the association between incarceration and population health is not driven by implausible age-specific mortality rates.

Despite these interesting results, the analysis herein nonetheless has five limitations. First, and maybe most importantly, I lack an exogenous shock in incarceration, and as such, despite the extensive robustness checks I conducted, it is unclear whether any of the associations herein are causal. To remedy this problem, future research might exploit exogenous shocks in incarceration to provide causal estimates (Drago, Galbiati, and Vertova, 2009; Johnson and Raphael, 2012; Levitt, 1996). Second, although some macro-level research on the consequences of incarceration for population health focuses on both the immediate and lingering consequences of shifts in the incarceration rate for population health (Johnson and Raphael, 2009), the current analysis was only able to focus on the immediate effects of shifts in incarceration and, thus, likely underestimates these effects. Third, the analysis focuses exclusively on the incarceration–population-health association in developed democracies, and it would be interesting to know what this association looks like in other types of countries. Fourth, as mentioned, there is likely substantial heterogeneity among the other 20 developed democracies included in this analysis in terms of consequences of incarceration, and by lumping these different nations together, this analyses ignores such heterogeneity. Fifth, it may be the case that this distinctive relationship is driven not by anything unique about the United States except for its high rate of incarceration and large annual increases in the incarceration rate. In a similar vein, I modeled differences in incarceration rates as the key explanatory variable, but it could be the case that the type of incarceration regime—including, for instance, conditions of confinement—may drive the association. Of all the avenues for future research in this area to consider, this is perhaps the most likely to bear fruit as understanding whether the effects of incarceration in the United States are distinctively detrimental because of the high starting rate of incarceration in the United States, the large annual increases in incarceration in the United States, the conditions of confinement in the United States, or something else entirely are vital for considering how to minimize the macro-level consequences of mass incarceration for health disparities within the United States and between the United States and other developed nations.

Limitations aside, this article has several important implications. For scholars interested in population health, it suggests that increases in incarceration could possibly explain part of the lagging improvements in population health in the United States relative to other developed democracies. For scholars interested in incarceration, the implications are maybe even more important. For those of us who spend our careers thinking about prisons and jails and the people who occupy them, we tend to think of the United States as unique in terms of its rate of incarceration—and the concentration of incarceration among young, minority men living in neighborhoods of concentrated disadvantage (Garland, 2001; Loeffler and Sampson, 2010). Yet the findings from this article indicate that the United States is an outlier not just in terms of its rate of incarceration but also in terms of how incarceration affects the health and well-being of its society, indicating extremity not on one level, as often suggested, but on two. As mentioned, the next phase of this research must show what is driving these differential effects.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher's web site:

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